EXHIBIT 1

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Report on Drospirenone Containing Combined Oral Contraceptives (COCs)

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Introduction

I have been asked to issue a report concerning the pharmacokinetic and pharmacodynamic properties of the brand of combined oral contraceptives (OCs or COCs) containing Drospirenone (DRSP) as the progestogenic agent in combination with ethinyl estradiol (EE), the synthetic estrogen component of both the Yasmin and Yaz products manufactured and sold by Bayer Healthcare Pharmaceuticals, Inc., or "Bayer".

This paper sets forth, among other things, my opinions concerning the nature and actions of DRSP when co-administered with EE in either one of two preparations (Yasmin/Yaz) that are currently marketed and sold for contraception. All of the opinions stated within this report are formulated and made with a reasonable degree of pharmacological and scientific certainty.

My opinions are based upon a combination of materials, including those that have been provided to me by counsel as well as materials that I have obtained during the course of my own research. The materials I have reviewed include multiple clinical study reports conducted of both the Yasmin and Yaz formulations during clinical trials, the relevant published literature, the product labeling, including the labeling for other hormonal contraceptive products, deposition testimony, and internal/external Bayer communications. In addition, I rely upon my own education, training and laboratory research in the relevant fields, including research that I have conducted with estrogens and with regard to the Renin-Aldosterone-Angiotensin System (RAAS), a subject on which I have been published. A reference and general bibliography of literature reviewed and considered by me in formulating my opinions are attached at the end of this report.

Nature of Estrogens

An adverse effect of estrogens used as oral contraceptive agents is thrombosis, which is inclusive of venous thromboembolism (VTE; deep vein thrombosis and pulmonary embolism) and arterial thromboembolism (ATE; stroke and myocardial infarction). Estrogens, including EE, are known to result in liver activation resulting in increases in the plasma concentration of many proteins of hepatic origin, including: sex-hormone binding globulin (SHBG), corticosteroid binding globulin (CBG), thyroxin binding globulin and angiotensinogen (Goodman and Gilman textbook of Pharmacology). Estrogen has effects on the liver to not only stimulate the synthesis of coagulation (clotting) factors such as factors VII, VIII, X and fibrinogen, but also to lower the levels of anticoagulant factors such as antithrombin III, thus resulting in a net prothrombotic effect (Vandenbroucke, 2001).

Relationship of Combined Oral Contraceptives to Thrombosis

The known action of estrogens to cause thrombosis has been reduced with the advent of combined oral contraceptives (COCs) possessing reduced doses of estrogens plus a progestogen. COCs containing a second generation progestogen (i.e., levonorgestrel) co-administered with

lower doses of EE, (i.e., < 50μg EE), have reduced adverse events relative to first generation COCs. The COCs containing third generation progestogens (i.e., norgestimate, desogestrel) were developed in the hope of further lowering adverse cardiovascular effects. However, studies have shown that use of low-dose COCs with a third-generation progestogen carries a two-fold higher risk of venous thrombosis than the previous generation of COCs (Bloemenkamp, 1995; Jick, 1995; Kemmeren, 2001; Fleischer, 2009) (Table 1). Consistent with this, it has been found that COCs containing third generation progestogens cause higher activated protein C (APC) resistance (Rosing, 1997) and higher SHBG levels (Odlind, 2002; van Rooijen, 2004, Fleischer, 2009) than second generation COCs. Based on these findings, the use of 3rd generation COCs has been the subject of much controversy and debate. In Europe the EMEA has adopted a "Position Statement" accepting the higher risk association with third-generation COCs. (Breitfeld Exhibit 1). In the United States, the label warns of an approximate two-fold increase in risk associated with desogestrel containing OCs, i.e., Mircette.[1]

Product Development and Description of DRSP-containing OCs

Drospirenone is the progestogen found in two COC preparations: Yaz, which contains 20 μg EE and 3 mg drospirenone, and Yasmin, which contains 30 μg EE and 3 mg of drospirenone. Yasmin is administered in a regimen of 21 days on active pill followed by 7 days of placebo. In contrast, Yaz is administered on a 24/4 day regimen, meaning there are three additional days of exposure to hormones.

In addition to contraception, Yaz has been approved by Food and Drug Administration (FDA) for the treatment of premenstrual dysphoric disorder (PMDD). (See Yaz/Yasmin labels, April 2010; see also, Pearlstein, 2005; Yonkers, 2005) and acne vulgaris (Harper, 2009). Drospirenone has progestational, antimineralocorticoid, and antiandrogenic activity (Oelkers, 1991; Oelkers, 1995; Krattenmacher, 2000) (Table 2). The antimineralocorticoid potency of drospirenone in rat and man is almost eight times as much as that of spironolactone (Muhn, 1995). Because drospirenone has antimineralocorticoid activity, it should not be used in patients who are receiving agents that may increase serum potassium concentration (i.e., ACE inhibitors, AT_1 receptor antagonists, potassium sparing diuretics, heparin, MR antagonists or NSAIDs).

Yasmin was first introduced in Europe in 2000 and was approved for sale by the FDA in 2001. (See Yasmin label, 4/2010). Yaz was approved first in the United States in 2006 and as stated above is also approved for acne and PMDD treatment. Shortly after the launch of Yasmin in Europe, reports of VTE in conjunction with ingestion of Yasmin surfaced and reports continued over the next several years about the safety of the DRSP containing OCs. Following the launch of Yasmin, Bayer was required by the regulatory authorities in both Europe and the U.S. to conduct post-marketing surveillance of the Yasmin product to assess its safety profile post-launch. In Europe, the study conducted was the European Active Surveillance (EURAS) study for which final results were published (Dinger, et al.) in 2007. The INGENIX study (Seeger, 2007) was initially designed to assess hyperkalemia events due to the potential for spironolactone analog to cause high levels of potassium. The latter study protocol was later

[1] In Europe, two different types of third-generation progestins are marketed in combination with EE: Desogestrel and Gestodene.. (See, e.g., Femodette label, a COC manufactured by Bayer at EMC website: http://www.medicines.org.uk/emc/medicine/2542/SPC/femodette/)

revised in accordance with FDA discussions so as to capture VTEs as well. The results of these earlier epidemiology studies for which final results were released in 2007, showed no increased risk of VTE with drospirenone-containing COCs compared with patients receiving 2nd generation COCs. However, subsequent reports appearing in the British Medical Journal indicated an increased risk of VTE with drospirenone-containing COCs when compared to 2nd generation COCs (Lidegaard, 2009; Van Hylckama Vlieg, 2009) (See also Table 1 attached).

More recently, two separate epidemiological studies looking at two different databases, one in the U.S. and the other in the United Kingdom, noted a respective 2-fold and 3-fold increase in VTE risk with drospirenone-containing COCs when compared with 2nd generation COCs (Jick, 2011; Parkin, 2011).

Although the epidemiology studies to date appear to have studied the Yasmin formulation (i.e., 30 μg EE/3mg DRSP) in comparing the risk of VTEs as against LNG-containing COCs, the present FDA-approved labels in the U.S. for each of these products references four epidemiology studies that were available when the label was revised (Lidegaard, 2009; Van Hylckama Vlieg, 2009; Dinger, 2007; and Seeger, 2007), without distinguishing between the two products. In addition, the Summary of Product Characteristics (SPC) in Europe for both Yaz and Yasmin reflect the current state of epidemiology and do not distinguish between these two DRSP-containing OCs.[2]

Findings as described previously after the launch of Yasmin and later Yaz, have prompted investigators to question the safety of COCs containing drospirenone compared to other available COCs. (Lidegaard, 2009; Vlieg, 2009; Jick, 2011; Parkin, 2011). In examining this issue from a pharmacologic perspective, three general mechanisms should be considered. First, drospirenone likely sensitizes target tissues to the effects of estrogen (a pharmacodynamic interaction). Secondly, drospirenone possesses an intrinsic effect to promote thrombosis independent of an interaction with EE. Thirdly, drospirenone appears to increase the magnitude and the duration over time of the interaction of estrogen with its receptor (a pharmacokinetic interaction), that may increase estrogenic exposure to the woman regardless of the actual dose administered in the product.

1. Ability of Drospirenone to Sensitize the Effects of Estrogen:

Drospirenone lacks estrogenic activity, but unlike most other progestogens possesses antiandrogenic activity. (Table 2). The progestational activity of drospirenone is four times that of progesterone and in addition to its progestational activity drospirenone has antimineralocorticoid activity. Unlike other progestogens, drospirenone has antiandrogenic activity rather than androgenic activity (Muhn, 1995). Drospirenone possesses eight to ten times the antiandrogenic activity of spironolactone. This may have important implications regarding the pharmacodynamic effects of ethinyl estradiol with which it is co-administered. Androgens, in general, have an antiestrogen effect and in the early days of breast cancer therapy, androgens were used to combat estrogen sensitive carcinoma. Dihydrotestosterone has been shown to down

[2] SPC Yaz: http://www.medicines.ie/medicine/13973/SPC/Yaz/ June 2011; and http://www.medicines.org.uk/EMC/medicine/8777/SPC/Yasmin 13/06/2011.

regulate estrogen receptors in the pig uterus, which may explain its ability to antagonize estrogenic effects at that site (Cardenas, 2003).[3] Although not approved by the FDA, spironolactone is widely prescribed by dermatologists for the treatment of acne due to its antiandrogenic activity. Yaz (20 µg EE and 3 mg drospirenone) was approved by the FDA for the treatment of moderate acne (Harper, 2009). Two other COCs have been approved by the FDA to treat acne, and although they contain progestins with androgen-like effects, their net effect is antiandrogenic as the added EE increases hepatic synthesis of SHBG, which binds free testosterone making it unavailable to be converted to dihydrotestosterone or to bind to the androgen receptor. In contrast, drospirenone not only decreases free testosterone levels but also has intrinsic anti-androgen activity independent of the presence of EE.

a. <u>The Effects of Drosperinone on Sex Hormone Binding Globulin</u>

SHBG, also known as TeBG (testosterone-estrogen binding globulin), SSBG (sex steroid binding globulin), or SBP (sex steroid binding protein), is synthesized by liver cells and has a 7-day half-life in circulation. SHBG binds to both androgens and estrogens with a 10-fold higher affinity for dihydrotestosterone than estradiol. Elevated testosterone levels cause SHBG synthesis to decrease, while estrogen stimulates SHBG production. With regard to COCs, most progestogens possess androgenic potential and this may counteract the effect of estrogens to stimulate SHBG levels, such that COCs containing less androgenic progestogens may further enhance SHBG levels (Martinez, 2007). In terms of the pharmacodynamic effects of drospirenone-containing COCs, it has been found that they produce marked increases in SHBG. During treatment with Yasmin there is a five-fold increase in the plasma concentration of SHBG from 70 nmol/L to 350 nmol/L (Yasmin SPC). In some reports, the degree to which SHBG increases following drospirenone-containing COC dosage has been underestimated as the resulting levels of SHBG were off scale (e.g., AW06, where levels above 300 nmol/ml could not be measured on certain occasions for a majority of the study subjects).[4] These changes in SHBG levels are much greater than those seen in second and even third generation progestin containing pills. [Odlind, et al., 2002]. As the androgenic nature of the progestin component decreases, a consequent rise in SHBG levels are seen, suggesting greater estrogenicity (independent of the dose administered) and the potential for greater thrombus formation. [Martinez, et al.; 2007]. In fact, some studies have found that the greater the degree of increase in SHBG levels, the greater the risk of VTE. [Odlind, et al. 2002].

Consonant with the ability of drospirenone-containing COCs to markedly increase SHBG levels is their known ability to decrease free testosterone levels. SHBG binds to testosterone with high affinity and thus increases in SHBG levels can decrease free testosterone levels due to the

[3] Importantly, androgens are not exclusively present in males and estrogens are not exclusively present in females. Thus, females have some basal levels of androgens and males have some basal levels of estrogens. It has been shown that androgen levels are increased in postmenopausal women and in women with polycystic ovary syndrome, the incidence for which may be as high as 7% in women at reproductive age.

[4] According to researchers out of the Leiden University, SHBG increases with estrogen exposure, such as with COCs, and is also associated with increased resistance to Activated Protein C (APCr), which in turn is indicative of a prothrombotic effect. [van Vliet HA, Frolich M, Christena M, Rosing, J. et al.; 2005] I defer to the hematology experts who may be asked to comment on the significance of APCr in users of COCs, in particular DRSP-containing OCs for which this data have been reported. [See also, Tchaikovski, SN, Rosing, J., 2010.]

avidity with which it binds to testosterone. Thus, not only does drospirenone have antiandrogenic (androgen receptor blocking) activity but, in addition, it lowers free testosterone levels. This is the rationale behind combining androgenic progestins, such as DRSP and CPA, with EE for the treatment of acne vulgaris. Indeed, Bayer acknowledges in study Report No. A40196 (p. 161) that DRSP has effects in the body (4-5 fold increase in SHBG levels) that are similar to CPA, a progestin that is not sold in the United States.[5]

CPA like drospirenone-containing COCs, is considered a fourth generation progestogen and is also being used for the treatment of acne vulgaris (Tchaikovski, S.N., et al. 2010). As mentioned above, CPA also markedly increases SHBG levels, decreases free testosterone levels and has antiandrogen (androgen receptor blocking) activity (Table 2). Although the effects of drospirenone plateau with time, the levels of SHBG continue to increase. Changes in SHBG have been reported to indicate the balance between estrogenic and androgenic activities and thus reflect net estrogenicity (Martinez, 1999). SHBG is not only found in the circulating plasma but may also bind at cellular sites, another potential mechanism for increasing the total "estrogenicity" seen with lower androgenic progestins, and even more so with anti-androgenic progestins such as DRSP. What is clear is that there is a greater increase in SHBG with drospirenone-containing COCs as reflected in Bayer's own internal studies for both the Yasmin product (e.g., Study Report Nos. AW06, 9274), as well as for Yaz (e.g., A40196, A25083),[6] and reported in the medical literature.

2. Intrinsic Effects of Progestogen to promote Thrombosis independent of Estrogen

With regard to intrinsic effects, drospirenone is unique among progestogens in that it possesses antimineralocorticoid activity which can cause a diuretic/natriuretic effect and activation of the renin-angiotensin-aldosterone system. An effect of drospirenone on body weight was reported in three studies (Carranza-Lira, 2009). In subjects receiving 0.5 to 2 mg of drospirenone there was no change or a decrease in body weight, in a second study no change in body weight was observed among groups receiving drospirenone and EE. However, in a third study, significant decreases in body weight from baseline were observed with doses of drospirenone of 2 and 3 mg per day. The weight loss is likely due to the antimineralocorticoid effect of drospirenone and thus reflects its diuretic/natriuretic ability. (Oelkers, 1991; Oelkers, 1995). In addition, it is known that drospirenone can increase renin release (Oelkers, 1991; Oelkers, 1995) which promotes the formation of angiotensin II. The medical literature supports that angiotensin II has toxic effects on blood vessels, and like cigarette smoking and advancing age, act in concert with the prothrombotic effects of estrogen to cause venous thromboembolism.

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[5] Bayer manufactures Diane-35, a COC containing CPA, that is sold in Europe and is used for the treatment of acne.

[6] For example, in Study Report No. A40196 at Page 161 of that report, it states that the 400-500% increase in SHBG levels seen in the Yaz study subjects is similar to that seen with women exposed to OCs containing "other non-androgenic progestins" such as DRSP. CPA is a non-androgenic progestin which has been found to have the highest VTE risk according to epidemiological studies. (Lidegaard, 2009; Vlieg, 2009; See also Table 1).

3. Drospirenone Appears to Alter the Pharmacokinetics of EE

a. Drospirenone Pharmacokinetics

With regard to the pharmacokinetics of drospirenone-containing COCs, the results regarding drospirenone levels have been fairly consistent whereas the reported EE levels have been inconsistent and variable. Following oral administration, the bioavailability of drospirenone is about 76%. The mean peak serum concentration of drospirenone following a single-oral dose of 3 mg is 36.9 ng/ml at about 1.7 hours, and mean peak steady state concentrations are 78.7 to 87.5 ng/ml. Drospirenone concentrations in women with moderate renal impairment are about 37% higher. Drospirenone is about 97% bound to serum albumin (Krattenmacher, 2000) but does not bind to SHBG (Krattenmacher, 2000). The elimination half-life is about 30 hours for drospirenone which exceeds the dosing interval which is every 24 hours. Some preclinical reports have indicated that drospirenone, like EE, undergoes enterohepatic recirculation. This mechanism allows drugs to re-enter the body after they have been released into the bile and can lead to protracted drug effects both with respect to DRSP itself as well as the EE with which it is co-administered. This effect would be expected to be more pronounced with the Yaz formulation which, although it contains 10μg less of EE, is administered over 24 days with only 3 days of placebo.[7] Two major metabolites are formed from drospirenone in the body and both are pharmacologically inactive. At least twenty metabolites of drospirenone have been detected in the urine or feces (Krattenmacher, 2000).

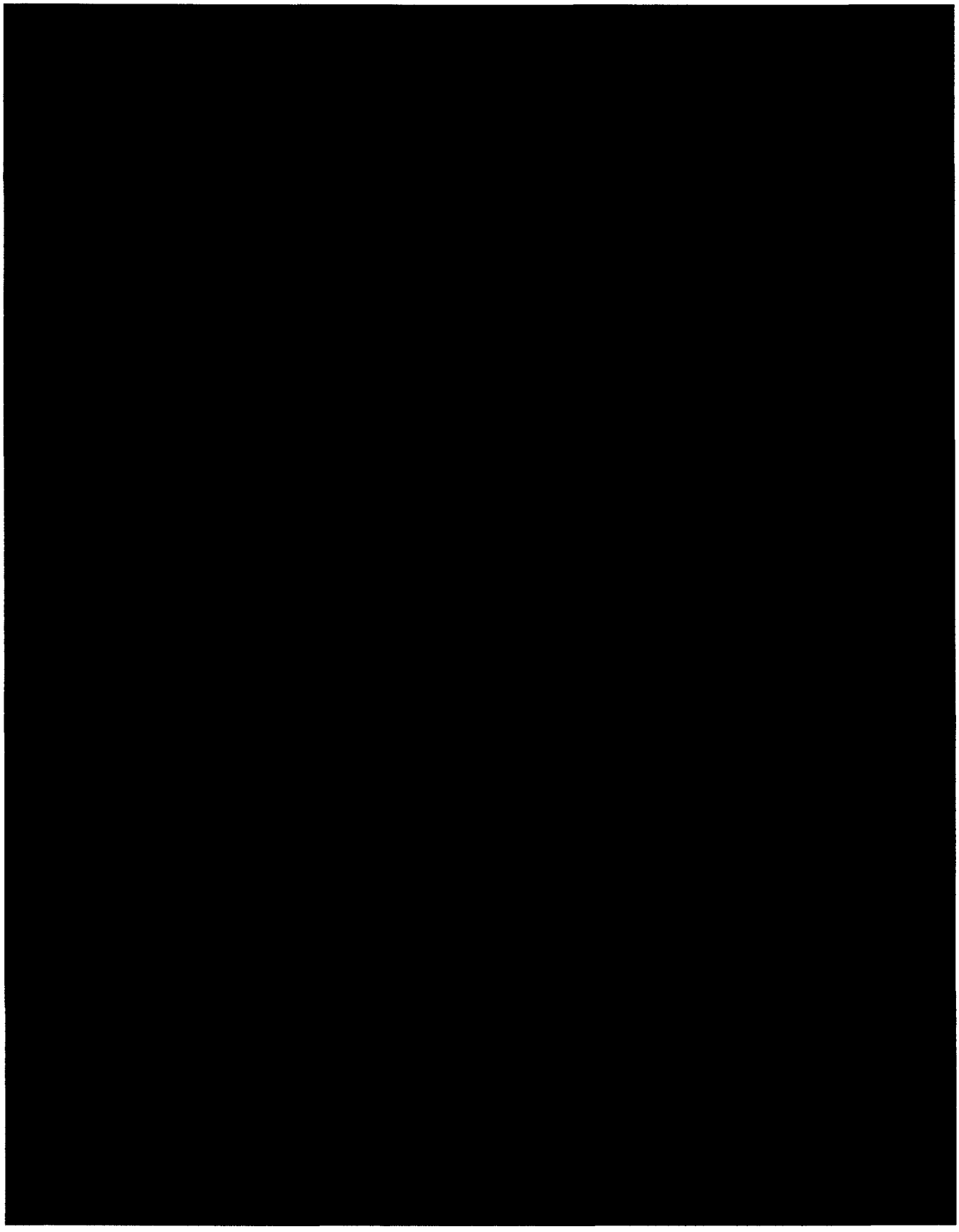
b. Ethinyl Estradiol Pharmacokinetics

Studies to investigate the effects of EE on drospirenone levels have found no effect. However, there have been no studies to investigate the effect of drospirenone on the plasma levels of EE that are achieved when it is co-administered with EE. Likewise, there is no information on whether metabolites of drospirenone affect EE levels that are achieved either with acute dosing, or more importantly, under chronic dosing conditions. As noted above, while Bayer tested the bioavailability of DRSP as affected by EE, it did not do the reverse testing relying instead on the "established, general scientific literature" on the absolute bioavailability of EE in COCs.[8]

[redacted]

[7] Dr. Joachim Marr, the core medical clinician at Bayer has testified that because of the long half life of DRSP coupled with the 24/4 regimen, the woman is never completely free of the influence of hormones from the Yaz Product. (See Deposition of Joachim Marr, 1/11/11, Tr. At 164-167).

[8] The Therapeutic Goods Administration (TGA) noted in its overview of Yasmin that this was a "significant deficiency" viz. product testing. [See Yasmin-Overview dated February 2001; Deposition of Joachim Marr, 4/18/11 Tr. At 60-61; Marr Exhibit W]



Conclusion and Opinion

Based on the above it is clear that Yaz and Yasmin behave differently from other COCs due the intrinsic differences in the property of the progestin that is combined with EE in the two formulations (Yasmin and Yaz) as described in detail above. Pharmacodynamically they exhibit effects consistent with increased estrogenicity as they lead to further enhancement of SHBG levels. There is a well-known association between increased SHBG levels and increased APC resistance, which is considered by some researchers to be predictive of VTE risk. [Van Vliet, 2005; Odlind, 2002]. This effect is also observed with CPA which, like drospirenone, possesses antiandrogenic activity. The measurement of EE levels in patients receiving drospirenone has yielded values that are highly variable and that can result in very high levels of estrogen exposure. With regard to the Yaz label, the levels of EE recorded in the label would be inconsistent with preventing pregnancy, and is in fact inconsistent with Bayer's own clinical studies reporting much higher levels of EE in the subjects tested. Together, these observations are consistent with epidemiological findings that indicate a high incidence of venous thromboembolism with the DRSP containing COCs over that of other preparations. (Table 1).

Given the totality of the available evidence, including the data from clinical trials, the product literature, the published medical literature, and the epidemiological evidence, it is my opinion with a reasonable degree of scientific and pharmacologic certainty that drospirenone-containing COCs produce a risk of VTE that is at least as high, and likely greater than the so-called third generation COCs or at least double that of Levonorgestrel containing products.

Attached to this Report is my curriculum vitae which sets forth my background, qualifications, education and training as well as articles published by me in the last ten years and prior. In a separate attachment, I disclose prior testimony at deposition and/or trial in the last four years as well as my schedule of professional fees.

I reserve my right to supplement these opinions further and to respond to any contrary opinion reports that may be submitted in this case.

Charles T. Stier, Jr., Ph.D.

Charles T. Stier, Jr., Ph.D.

Date: 7/28/2011

TABLES

Table 1 Relative Risk of Combined Oral Contraceptives

	MEGA Study [1]	Danish Cohort Study [2]
First Generation		
Norethisterone	3.9 (1.4-10.6)	
Second Generation		
Levonorgestrel	3.6 (2.9-4.6)	2.0 (1.75-2.34)
Third Generation		
Desogestrel	7.3 (5.3-10.0)	3.55 (3.30-3.83)
Gestodene	5.6 (3.7-3.8)	
Norgestimate	5.9 (1.7-21.0)	
Fourth Generation		
Drospirenone	6.3 (2.9-13.7)	4.00 (3.26-4.91)
Cyproterone Acetate	6.8 (4.7-10.0)	

[1] van Hylckama Vlieg, 2009
[2] Lidegaard, 2009

Table 2 Receptor Effects of Various Progestagens

Progestagen	Generation	Progesterone Receptor	Estrogen Receptor	Androgen Receptor	Mineralocorticoid Receptor
Drospirenone	IV	(+)	0	(-)	(-)
Cyproterone Acetate	IV	(+)	0	(-)	0
Desogestrel	III	(+)	0	0/(+)	0
Gestodene	III	(+)	0	0/(+)	0
Norgestimate	III	(+)	0	0/(+)	0
Levonorgestrel	II	(+)	0	0/(+)	0

(+) = angonist 0 = no effect (-) = antagonist

Table 3 Pharmacokinetic Comparisons

		LoLoestrin (1) 10 µg	Yaz (2) 20 µg	Yasmin (3) 30 µg
Cycle 1	Cmax	50.9 (27)	32.8 (45)	53.5 (27)
Day 1	AUC 0-24	389.9 (27)	108 (52)	280.3 (87)
Cycle 1	Cmax	71.3 (33)	45.1 (35)	92.1 (35)
Day 21	AUC 0-24	621.3 (41)	220 (57)	461.3 (94)

(%CV) = percent coefficient of variation = standard deviation divided by the mean

(1) LoLoestrin Package Insert

(2) Yaz Package Insert

(3) Yasmin Package Insert

TABLE 4 Mean AUC(0-24)*

Study Subject #	Occasion 1 (FE level)	Study Subject #	Occasion 2 (FE level)
4155	320.3	3720	367.17
3699	330.85	3691	437.48
3454	340.96	3854	442.33
3996	359.5	4244	445.43
3026	406.52	3056	456.24
3829	2007.4	3414	2068.4
4271	2080.5	4112	2156.5
4342	2245.8	4291	2389.1
4345	2321.9	4317	2394.9
3953	2509.9	3254	3203.6

* Blode Exhibit 18 (Study Report No. A47605 (Protocol No. 308683)).

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Estrosep Package Insert (current Label)

Femodette Summary of Product Characteristics; European Medicines Commission (EMC): http://www.medicines.org.uk/emc/medicine/2542/SPC/femodette/

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Materials Provided to Expert Dr. Charles Stier

In addition to the materials specifically referenced in my report, the other materials I have considered are:

Litigation Documents/Responses:

1. Interrogatory Responses designating clinical and pre-clinical (animal) study reports
2. YAZ – Amended ATTACHMENT A – MDL-PA INTERRS SET 1 – CLINICAL.xls
3. YAZ – ATTACHEMENT A (Spreadsheet)– INDEX TO APPENDICES TO CLINICAL STUDIES.xls
4. YAZ – AMENDED ATTACHMENT A (Spreadsheet) – MDL-PA INTERRS SET 1 – PRECLIN.xls
5. Bayer "Lunch and Learn" DVD

FDA Filings:

6. Yasmin NDA 21-098 and Approval Package
7. YAZ 21-676 NDA and Approval Package
8. YAZ 21-873 NDA and Approval Package
9. YAZ 22-045 NDA and Approval Package
10. Clinical Study Reports:
11. Clinical Study 4417
12. Clinical Study 5824
13. Clinical Study 6737
14. Clinical Study 6961
15. Clinical Study 7214
16. Clinical Study 7215
17. Clinical Study 8036
18. Clinical Study 8235
19. Clinical Study 8256
20. Clinical Study 8644
21. Clinical Study 9274, with corresponding appendices, tables, and graphs
22. Clinical Study 9370, with corresponding appendices, tables, and graphs
23. Clinical Study 9371
24. Clinical Study 9482
25. Clinical Study 9692
26. Clinical Study 9693, with corresponding appendices, tables, and graphs
27. Clinical Study 9776, with corresponding appendices, tables, and graphs
28. Clinical Study 98180
29. Clinical Study 9970

30. Clinical Study A00824
31. Clinical Study A01222
32. Clinical Study A01438
33. Clinical Study A03161
34. Clinical Study A03328, with corresponding appendices, tables, and graphs
35. Clinical Study A03773
36. Clinical Study A06872
37. Clinical Study A07051
38. Clinical Study A07148
39. Clinical Study A07545
40. Clinical Study A07735
41. Clinical Study A09151, with corresponding appendices, tables, and graphs
42. Clinical Study A09372
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52. Clinical Study A21566
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54. Clinical Study A25152
55. Clinical Study A25848
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57. Clinical Study A26560
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59. Clinical Study A29551
60. Clinical Study A30713
61. Clinical Study A32276
62. Clinical Study A32630
63. Clinical Study A40196, with corresponding appendices, tables, and graphs
64. Clinical Study A41540
65. Clinical Study A41541
66. Clinical Study A43598
67. Clinical Study A470, with corresponding appendices, tables, and graphs
68. Clinical Study A48046
69. Clinical Study A49202

70. Clinical Study A733
71. Clinical Study A892, with corresponding appendices, tables, and graphs
72. Clinical Study A951, with corresponding appendices, tables, and graphs
73. Clinical Study AE91
74. Clinical Study AG44
75. Clinical Study AH37
76. Clinical Study AI51
77. Clinical Study AI98
78. Clinical Study AI99
79. Clinical Study AJ06
80. Clinical Study AL84
81. Clinical Study AM80
82. Clinical Study AM90
83. Clinical Study AM91
84. Clinical Study AV29, with corresponding appendices, tables, and graphs
85. Clinical Study AW06, with corresponding appendices, tables, and graphs
86. Clinical Study AW45
87. Clinical Study B277
88. Clinical Study B283
89. Clinical Study B601
90. Clinical Study B682
91. Clinical Study B862
92. Clinical Study B990
93. Clinical Study BD09

Spreadsheet: Clinical Study Reports/Corresponding Published Articles

Published Articles related to clinical study reports:

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Deposition Exhibits & Deposition Testimony and Related Materials:

195. Declaration of Hartmut Blode (BSPYAZ020380583)
196. Deposition testimony of Dr. Joachim Marr
197. Deposition testimony of Dr. Ilka Schellschmidt
198. Deposition testimony of Hartmut Blode
199. Deposition testimony of Brietfeld
200. Dr. Blode Exhibits 1 – 31 (PERSONAL DEPOSITION)
201. Dr. BLode Exhibits 1- 5 (Rule 30(b)(6) deposition
202. Fiedler Exhibit 5
203. Foegh Exhibit 11
204. Lynen Exhibit 15
205. Lynen Exhibit 16
206. Plaintiffs 655

Other Materials:

207. TGA Clinical Evaluation Report- Yasmin (2000) (BSPYAZ010891319*[11])
208. Grimes et al (2010) Editors Corner-Surrogate End Points – "Surrogate End Points in Womens health research: science, protoscience, and pseudoscience" (BHCPYAZ016434134)
209. Email re published articles (BHCPYAZ016434128)
210. Grimes Surrogate Endpoints Article (BHCPYAZ016434129) – "Surrogate End Points in Clinical Research: Hazardous to your health".
211. "Sex hormone-binding globulin: an adequate surrogate marker for venous thromboembolism in women using new hormonal contraceptives"– Letter to Editor, Contraception 79 (2009)
212. Shakir (DSRU) Letter to British Regulatory authorities re: Preliminary results of PEM study; Feb. 19, 2004 (BHCPYAZ00283354 – 28pgs)

[1] References to Bates stamped documents are to the first page of each complete document, if not otherwise identified by title or exhibit number.

213. YAZ Clinical Pharmacology Label – BHCPYAZ013324558
214. Yasmin Original Label (2001)
215. YAZ 24/4 (2006 Original Label)
216. Yasmin Revised label (April 2010)
217. YAZ Revised Label (April 2010)
218. EU Press Release March 26, 2010 re: Label Change
219. YAZ Revised Label March 2011
220. Mircette Label
221. Desogen Label
222. Cyclessa label
223. Lidegaard Re-Analysis Report and tables dated March 29th 2011.
224. Shapiro Appraisal – BSPYAZ020502208
225. Shapiro Comment – BSPYAZ020502218
226. BSPYAZ010891487 ADEC Meeting
227. TGA Overview of Yasmin – BSPYAZ010891389
228. EMA – PhVWP Monthly Report (May 2011)
229. FDA.com article – "FDA Drug Safety Communication: Safety Review of possible increased risk of blood clots with birth control pills containing drospirenone" - http://www.fda.gov/Drugs/DrugSafety/ucm257164.htm
230. Estrogen & Progestin PK Interaction Powerpoint June 2010 – BSPYAZ004065576
231. Summary Product Characteristics (EU) Yasmin 6/2011
232. Summary Product Characteristics (EU) Yaz 6/2011

CURRICULUM VITAE

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Personal

Born July 27, 1951 - Long Island, New York
Married, Two children

Education and Academic Honors

B.S. State University of New York, Stony Brook, New York, 1973, Chemistry, New York State Regents Scholarship 1969-1973

Ph.D. College of Physicians and Surgeons of Columbia University, New York, New York, 1978, Pharmacology, Advisor: Dr. Wilbur H. Sawyer
Columbia University Fellowship, 1973-1978

Post Graduate Training

NIH Postdoctoral Fellowship
Department of Medicine, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, 1978-1981. Specialty: Renal Physiology. Advisors: Carl W. Gottschalk, M.D. and William J. Arendshorst, Ph.D.

Academic Appointments

1990-present Associate Professor, Department of Pharmacology, New York Medical College, Valhalla, New York

1981-1990 Assistant Professor, Department of Pharmacology, New York Medical College, Valhalla, New York

1981-1990 Adjunct Assistant Professor, Department of Medicine, New York Medical College, Valhalla, New York

1981 Instructor, Department of Physiology, University of North Carolina, Chapel Hill, North Carolina

Professional Society Memberships

American Society of Nephrology (9/1/80-present)
American Heart Association, Council on Kidney in Cardiovascular Disease (7/1/82-present)
Eastern Hypertension Society (9/1/82-present)
The New York Society of Nephrology (9/1/84-present)
American Federation for Clinical Research (7/1/86-6/30/1994)
American Society of Hypertension (11/1/89-present)
American Heart Association, Council on High Blood Pressure (9/1/90-present)
American Society of Pharmacology and Experimental Therapeutics (1/1/91-present)
Fellow, American Heart Association Council for High Blood Pressure Research (1998-present)

Extramural Activities

SBIR Study Section Neurodegenerative Diseases,
July 28-29, 1997, Silver Springs, Washington D.C.
Special Emphasis Panel for review of NIH grant applications (April, 2000)
Editorial Board: Cardiovascular Drug Reviews (1999-present)
Editorial Board: American Journal of Hypertension (2000-2005)
Editorial Board: Current Hypertension Reviews (2004-present)
Co-Executive Editor: American Journal of Hypertension (2005-present)
AHA Northeast One Study Group (Integrated Cardiovascular Biology (2007-2008)

Intramural Administrative Activities

Member, Medical School Admissions Committee (1982-1985).
Member, Graduate Faculty Council (1984-present)
Member, Radioisotope Committee (1985-1992)
Director of the Graduate Course in Cardiovascular Pharmacology (Pharmacology 562.2) (1985-1986, 1987-1988, 1989-1990, 1992-1993, 1994, 1996, 1998, 2000, 2002)
Member, Graduate Program Directors Committee (1986-present)
Co-Director of the Graduate Program in Pharmacology (1986-present)
Enrollment Committee (1990-present)
Graduate Program Director for the Masters Program (1990-present)
Course Director, Pharmacology 521.4 Fall and Spring (1990-present)
Course Director, Pharmacology 590.1 Masters Seminar and Thesis (1990-present)
New York Medical College Faculty Senate (1990-present)
Library Search Committee, Medical Library Director (1991-1992)
Chairman, Ad hoc Committee on Graduate School Policy and Procedure (1993)
NYMC Task Force on Outcomes and Institutional Effectiveness (1994)
Course Director, Pharmacology 500.2 Fundamentals of Pharmacology (1994-present)
Member, Alternate Pathways Committee (1994-present)
Member, Academic Standards and Credentials Committee (1994-2006)
Member, Course Evaluation Committee (1996)
Member, Pre-professional M.S. Program Advisory Committee (1996-present)
Member, Curriculum Committee (1996-present)
Member, Intellectual Property Committee (1998-present)
Graduate School of Basic Medical Science Self Study Task Force (2001-2002)
Basic Medical Sciences Program Advisory Committee (2002-present)
Executive Committee, New York Medical College Faculty Senate (2002-2006)
Outstanding Faculty Award in the Graduate School of Basic Medical Sciences (2004)
Medical School Admissions Committee, New York Medical College (2004-present)

Chairman, Academic Standards and Credentials Committee (2006-present)
Executive Committee, New York Medical College Faculty Senate (2009-present)

Grants Held: Charles T. Stier, Jr./Principal Investigator

1. American Heart Association, North Carolina Affiliate Grant-in-Aid. Effect of dopamine on glomerular ultrafiltration dynamics. 7/1/81-6/30/82.

2. New York Medical College Grant-in-Aid. Intrarenal Formation of Serotonin from its Amino Acid Precursors. 8/1/82-7/31/83.

3. Pharmaceutical Manufacturers Association Foundation Research Starter Grant. Role of thromboxane in water balance, renal hemodynamics and development of hypertension spontaneously hypertensive rats (SHR). 1/1/83-12/31/84.

4. American Heart Association, National Center. Thromboxane, fluid balance and renal function in hypertension.

5. National Institutes of Health. Role of eicosanoids in renin release and renal function.

6. National Institutes of Health. Thromboxane in severe hypertension. 12/1/85-11/30/88.

7. E.R. Squibb and Sons, Inc. SQ 29,852: Protective action in salt-loaded stroke-prone SHR. 1/1/89-12/31/89.

8. American Heart Association, National Center. Stroke prevention and reversal of renal dysfunction in SHRSP. 7/1/89-6/30/92.

9. National Institutes of Health (NHLBI). Prevention of stroke and kidney dysfunction by ACE inhibition. 7/1/89-6/30/92.

10. Merck Sharp and Dohme Research Laboratories. Medical Grants Program. 10/1/89-6/30/90.

11. Marion Laboratories. Beraprost sodium studies in stroke-prone SHR. 12/1/90-6/30/91.

12. DuPont. DuP-753 studies in stroke-prone SHR. 7/1/91-12/1/91.

13. UpJohn Laboratories. Study of 21-aminosteroids in stroke-prone SHR. 12/1/91-11/30/92.

14. American Heart Association, New York State Affiliate, Inc. Angiotensin and vascular damage in stroke-prone spontaneously hypertensive rats. 7/1/92-6/30/95.

15. HBI Henri Beaufour Institute, USA, Inc. Effect of lanreotide on diuresis and other renal functions in rats. 4/1/93-9/30/93.

16. New York Medical College Special Initiative Research Grant. Vascular Protection by Estrogen in Female Stroke-Prone Rats 7/1/93-6/30/94.

17. Merck Sharp and Dohme Research Laboratories. Medical Grants Program. Simvastatin in stroke-prone SHR. 9/1/93 - 8/31/94.

18. National Institutes of Health (NHLBI) Prevention of stroke and kidney dysfunction by ACE inhibition. 12/1/93 - 11/30/97.

19. ASPET Committee on Educational Affairs. Individual Fellowship Program for Terence Litavec 6/1/94 - 8/15/94.

20. HBI Henri Beaufour Institute, USA, Inc. Effect of lanreotide on diuresis and other renal functions in rats 1995-1995. (SUPPLEMENT).

21. G.D. Searle & Co. Epoxymexrenone (Eplerenone) studies in stroke-prone SHR. 10/1/97 - 6/30/98.

22. American Heart Association, New York State Affiliate, Inc. Role of mineralocorticoids in the vascular pathology of stroke-prone hypertensive rats. 7/1/98-6/30/01.

23. National Institutes of Health (NHLBI) Prevention of stroke and kidney dysfunction by ACE inhibition. 9/30/99 - 8/31/03.

24. G.D. Searle & Co. Studies with eplerenone in rats with cyclosporin-induced nephrotoxicity. 3/01/2000 - 2/28/2001.

25. G.D. Searle & Co. Studies with eplerenone in rats with 5/6 nephrectomy. 3/01/2000 - 2/28/2001.

26. Bristol-Meyers Squibb Studies with omapatrilat in stroke-prone spontaneously hypertensive rats. 7/01/2000 - 6/30/2001.

27. BioNebraska. Studies with glucagon-like peptide-1 in stroke-prone spontaneously hypertensive rats. 10/01/2000 - 9/30/2001.

28. Pharmacia. Eplerenone and reactive oxygen species. 01/01/2001 - 12/31/2001.

29. National Institutes of Health (NHLBI) Prevention of stroke and kidney dysfunction by ACE inhibition. Minority Postdoctoral Supplement for Dr. Alafuro Ourene 01/01/2001 - 8/31/2003.

30. Novartis. Impact of aldosterone synthase inhibition on the development of vascular pathology in stroke-prone spontaneously hypertensive rats. 10/01/2002 - 12/31/2003.

31. Charles River Laboratories. Studies on Charles River stroke-prone spontaneously hypertensive rats. 07/01/2004 - 06/30/2005.

32. Novartis. Reversal of end-organ damage by Diovan. 10/01/2004 - 12/31/2006.

33. New York Medical College Intramural Research Support Program. EETs in salt-sensitive end-organ damage in stroke-prone spontaneously hypertensive rats. 12/01/2005 - 11/30/2006.

34. Charles River Laboratories. Studies on Charles River obese spontaneously hypertensive rats (SHROB). 07/01/2008 - 06/30/2009 (Submitted).

35. National Institutes of Health (NHLBI). Salt-sensitive end-organ damage in severe hypertension. 04/01/2009 - 03/31/2014 (Submitted).

36. American Heart Association Founders Affiliate Grant-in-Aid. Protective role of EETs against salt-sensitive target-organ damage. 07/01/2009 - 06/30/2012 (Submitted).

37. New York Medical College Intramural Research Support Program. Niacin treatment for metabolic

syndrome in rats. 2/01/2009 - 01/31/2010.

Publications

1. Eakins, K.E., C. Stier, P. Bhattacherjee and L.M. Greenbaum. Actions and interactions of bradykinin, prostaglandins and nonsteroidal anti-inflammatory agents on the eye. Inflammation 1:117-125, 1975.

2. Manning, M., J. Lowbridge, C.T. Stier, Jr., J. Haldar and W.H. Sawyer. [1-Deaminopenicillamine, 4-valine]-8-D-arginine-vasopressin, a highly potent inhibitor of the vasopressor response to arginine-vasopressin. J. Med. Chem. 20:1228-1230, 1977.

3. Stier, C.T., Jr., M. Manning and W.H. Sawyer. Natriuretic effect of [7-glycine]-oxytocin in the presence of diuretic agents in conscious rats. J. Pharmacol. Exp. Ther. 212:412-417, 1980.

4. Stier, C.T., Jr., M. Manning and W.H. Sawyer. Effects of structural changes on the natriuretic activity of oxytocin analogs in conscious rats. Proc. Soc. Exp. Biol. Med. 164:167-172, 1980.

5. Stier, C.T., Jr., E.A. Cowden and M.E.M. Allison. Effects of bromocriptine on single nephron and whole kidney function in rats. J. Pharmacol. Exp. Ther. 220:366-370, 1982.

6. Stier, C.T., Jr., G. McKendall and H.D. Itskovitz. Serotonin formation in non-blood-perfused rat kidneys. J. Pharmacol. Exp. Ther. 228:53-56, 1984.

7. Dilley, J.R., C.T. Stier, Jr. and W.J. Arendshorst. Abnormalities in glomerular function in rats developing spontaneous hypertension. Am. J. Physiol. 246:F12-F20, 1984.

8. Stier, C.T., Jr., E.A. Cowden, H.G. Friesen and M.E.M. Allison. Prolactin and the rat kidney: a clearance and micropuncture study. Endocrinology 115:362-367, 1984.

9. Stier, C.T., Jr. and H.D. Itskovitz. Formation of serotonin by rat kidneys in vivo. Proc. Soc. Exp. Biol. Med. 180:550-557, 1985.

10. Lam, B.K., C.T. Stier, Jr., N.C. Wynn and H.D. Itskovitz. Dopamine and L-dopa potentiation of renal pressor responses to norepinephrine in isolated perfused rat kidneys. J. Cardiovasc. Pharmacol. 8:554-558, 1986.

11. Stier, C.T., Jr. and H.D. Itskovitz. Renal calcium metabolism and diuretics. Ann. Rev. Pharmacol. Toxicol. 26:101-116, 1986.

12. Stier, C.T., Jr., T.F. Brewer, L. Dick, N.C. Wynn and H.D. Itskovitz. Formation of biogenic amines by isolated kidneys of spontaneously hypertensive rats. Life Sciences 38:7-14, 1986.

13. Stier, C.T., Jr., L.J. Roberts, II and P.Y-K. Wong. Renal response to 9α,11β-prostaglandin F_2 in the rat. J. Pharmacol. Exp. Ther. 243:487-491, 1987.

14. Stier, C.T., Jr. and H.D. Itskovitz. Thromboxane A_2 and the development of hypertension in spontaneously hypertensive rats. Eur. J. Pharmacol. 146:129-135, 1988.

15. Stier, C.T., Jr. and E.G. Spokas. Thromboxane A_2 involvement in hypertension. Cardiovascular Reviews and Reports 9:32-38, 1988.

16. Stier, C.T., Jr., I.F. Benter and S. Levine. Thromboxane A_2 in severe hypertension and stroke in stroke-prone spontaneously hypertensive rats. Stroke 19:1145-1150, 1988.

17. Itskovitz, H.D., Y-H Chen and C.T. Stier, Jr. Reciprocal renal effects of dopamine and 5-hydroxytryptamine formed within the rat kidney. Clin. Sci. 75:503-507, 1988.

18. Stier, C.T., Jr., I.F. Benter, S. Ahmad, H. Zuo, N. Selig, S. Roethel, S. Levine and H.D. Itskovitz. Enalapril prevents stroke and kidney dysfunction in salt-loaded stroke-prone spontaneously hypertensive rats. Hypertension 13:115-121, 1989.

19. Itskovitz, H.D., J.L. Werber, A.M. Sheridan, T.F. Brewer and C.T. Stier, Jr. 5-Hydroxytryptophan and carbidopa in spontaneously hypertensive rats. J. Hypertension 7:311-315, 1989.

20. Ding, X.R., C.T. Stier, Jr. and H.D. Itskovitz. Serotonin and 5-hydroxytryptophan on blood pressure and renal blood flow in anesthetized rats. The American Journal of the Medical Sciences 297:290-293, 1989.

21. Ding, X.R., C.T. Stier, Jr. and H.D. Itskovitz. Hemodynamic effect of norepinephrine and serotonin with and without indomethacin. Chinese Medical Journal 102:461-463, 1989.

22. Lin, L., M. Mistry, C.T. Stier, Jr. and A. Nasjletti. Role of prostanoids in renin-dependent and renin-independent hypertension. Hypertension 17:517-525, 1991.

23. Stier, C.T., Jr., G.J. Sim and S. Levine. Dietary arginine fails to protect against cerebrovascular damage in stroke-prone hypertensive rats. Brain Research 549:354-356, 1991.

24. Stier, C.T., Jr., P. Chander, W.H. Gutstein, S. Levine and H.D. Itskovitz. Therapeutic benefit of captopril in salt-loaded stroke-prone spontaneously hypertensive rats is independent of hypotensive effect. Am. J. Hypertens. 4:680-687, 1991.

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26. Stier, C.T., Jr., N. Selig and H.D. Itskovitz. Enhanced vasodilatory responses to bradykinin in stroke-prone spontaneously hypertensive rats. European J. Pharmacol. 210:217-219, 1992.

27. Munsiff, A.V., P.N. Chander, S. Levine and C.T. Stier, Jr. The lipoxygenase inhibitor phenidone protects against proteinuria and stroke in stroke-prone spontaneously hypertensive rats. Am. J. Hypertens. 5:56-63, 1992.

28. Stier, C.T., Jr., K. Mahboubi, V.A. DiPippo, S. Levine and P.N. Chander. The antiproteinuric action of enalapril in stroke-prone spontaneously hypertensive rats is unrelated to alterations in urinary prostaglandins. J. Pharmacol. Exp. Ther. 260:1410-1415, 1992.

29. Stier, C.T., Jr., P.Y-K. Wong and H.D. Itskovitz. Enhanced thromboxane formation by blood but not whole platelets from spontaneously hypertensive rats. Prostaglandins 43:533-544, 1992.

30. Stier, C.T., Jr., C.P. Quilley and J.C. McGiff. Endothelin-3 effects on renal function and prostanoid

release in the rat isolated kidney. J. Pharmacol. Exp. Ther. 262:252-256, 1992.

31. Stier, C.T., Jr., P.E. Ward and S. Ahmad. Hyperresponsiveness to bradykinin in stroke-prone spontaneously hypertensive rats in vivo. Pharmacology Communications 1:353-359, 1992.

32. Stier, C.T., Jr., L.A. Adler, S. Levine and P.N. Chander. Stroke prevention by losartan in stroke-prone spontaneously hypertensive rats. J. Hypertension 11 (suppl 3):S37-42, 1993.

33. Stier, C.T., Jr., H.D. Itskovitz and Y-H Chen. Urinary dopamine and sodium excretion in spontaneously hypertensive rats. Clin. and Exper. Hypertension 15:105-123, 1993.

34. Hernandez, N.E., J.S. MacDonall, C.T. Stier, Jr., A. Belmonte and S.E. Karpiak. GM1 ganglioside treatment of spontaneously hypertensive stroke prone rats. Experimental Neurology 126:95-100, 1994.

35. Stier, C. T., Jr. Diuretics In: Basic Pharmacology in Medicine, ed. by J.R. DiPalma, G.J. DiGregorio, E.J., Barbieri and A.P., Ferko, Medical Surveillance Inc., West Chester, New York, Chapter 31, pp. 445-467, 1994.

36. Chander, P.N. and C.T. Stier, Jr. Stroke-prone spontaneously hypertensive rats: role of the renin angiotensin system and nitric oxide. Proceedings of the Fifth Asian Pacific Congress of Nephrology 32:299-310, 1995.

37. Johnson, R.A., A. Belmonte, N. Fan, M. Levesa, A. Nasjletti and C.T. Stier, Jr. Effect of ifetroban, a thromboxane receptor antagonist, in stroke-prone spontaneously hypertensive rats. Clin. and Exper. Hypertension 18:171-188, 1996.

38. Fan, N., C.A. Powers and C.T. Stier, Jr. Lack of antidiuretic activity of lanreotide in the diabetes insipidus rat. J. Pharmacol. Exp. Ther. 276:875-881, 1996.

39. Stier, C.T., Jr., P.N. Chander, A. Belmonte, R.S. Inamdar and M. Mistry. Beneficical action of beraprost sodium, a prostacyclin analog, in stroke-prone rats. J. Cardiovasc. Pharmacol. 30:285-293, 1997.

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41. Rocha, R., P.N Chander, K. Khanna, A. Zuckerman and C.T. Stier, Jr. Mineralocorticoid blockade reduces vascular injury in stroke-prone hypertensive rats. Hypertension. 31:451-458, 1998.

42. Fink, J., N. Y-T. Fan, L. Rosenfeld and C.T. Stier, Jr. Contribution of endothelin to the acute pressor response of L-NAME in stroke-prone spontaneously hypertensive rats. J. Cardiovasc. Pharmacol. 31:618-622, 1998.

43. Stier, C.T., Jr., P.N. Chander, A. Zuckerman and R. Rocha. Non-epithelial effects of aldosterone. Current Opinion in Endocrinology and Diabetes. 5(3):211-216, 1998.

44. Rocha, R., P.N. Chander, A. Zuckerman and C.T. Stier, Jr. Role of aldosterone in renal vascular injury in stroke-prone hypertensive rats. Hypertension. 33:232-237, 1999.

45. Mathew, R., N. Y-T. Fan, N. Yuan, P.N. Chander, M.H. Gewitz and C.T. Stier, Jr. Inhibition of

NOS enhances pulmonary vascular changes in stroke-prone spontaneously hypertensive rats. Am. J. Physiol. Lung Cell Mol. Physiol. 278: L81-89, 2000.

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111. Stier, C.T., Jr., G. Singh, R. Sepehrdad and P.N. Chander. Enhanced end-organ protection in the absence of increased plasma potassium with combined mineralocorticoid receptor (MR) antagonist and amiloride therapy. The FASEB Journal. 19(4) Part 1: A1188, 2005.

112. Chander, P.N., C.C.A. Chen, S.N. Masineni, C.A. Powers and C.T. Stier, Jr. Ovarian hormones partially protect against thrombotic angiopathy in aged female SHRSP independent of blood pressure. J Am Soc Nephrol. 16:697A, 2005.

113. Li, J., M.A. Carroll, S.N. Masineni and C.T. Stier, Jr. EETs excretion is decreased in response to high salt intake in stroke-prone spontaneously hypertensive rats. The 8th Annual Winter Eicosanoid Conference. Baltimore, Maryland, March 12-15, 2006.

114. Li, J., M.A. Carroll, S.N. Masineni and C.T. Stier, Jr. High salt intake decreases EET excretion in stroke-prone spontaneously hypertensive rats (SHRSP). FASEB J. 2006;20LB39-LB40.

115. Stier, C.T., C.C. Chen, C.B. Clifford, D.R. Owens and S.N. Masineni. High salt intake conditions stroke-prone spontaneously hypertensive rats to early stroke and renal damage as compared with their hypertensive and normotensive progenitor strains. J Clin Hypertens. 8(5): A39, 2006.

116. Stier, C.T., Jr., I.R. Hassan, S.N. Masineni, G.D. Singh, T.Y. Chun, J.H. Pratt and P.N. Chander. Cardiovascular consequences of long-term therapy with eplerenone in non-salt loaded stroke-prone hypertensive rats. 32nd Annual International Aldosterone Conference, Boston, Massachusetts, June 22 & 23, 2006. (Podium Presentation).

117. Schilt, N., C.T. Stier, Jr., L.I. Serova, S.N. Masineni and E.L. Sabban. Modulation of rat blood pressure, heart rate and locomotion during and subsequent to single and repeated immobilization stress: telemetric measurements. Neuroscience Meeting, 2006.

118. Stier, C.T., Jr., C.C.A. Chen, C.B. Clifford, D.R. Owens, S.N. Masineni. High-salt intake conditions Charles River stroke-prone spontaneously hypertensive rats to early stroke and renal damage. International Society of Hypertension, Abstract Book page 102, Fukuoka, Japan, October 15-19, 2006.

119. Stier, C.T., Jr., I.R. Hassan, S.N. Masineni, G.D. Singh, C.C.A. Chen and P.N. Chander Eplerenone therapy in the absence of high-salt intake affords cardiac protection in stroke-prone SHR. International Society of Hypertension Abstract Book page 406, Fukuoka, Japan, October 15-19, 2006.

120. Li, J., D. Karel, J.R. Falck, M.A. Carroll and C.T. Stier, Jr. The soluble epoxide hydrolase (sEH) inhibitor, AUDA, prevents the early salt-sensitive component of hypertension in stroke-prone spontaneously hypertensive rats (SHRSP). The FASEB Journal. 21(4) Part 1: A2222, 2007.

121. Sabban, E.L., N. Schilt, L.I. Serova and C.T. Stier, Jr. Acute and chronic cardiovascular and locomotive actions of ACTH and immobilization stress in rats: a telemetric study. Neuroscience Meeting 2007.

122. Li, J., M.A. Carroll, J.R. Falck and C.T. Stier, Jr. Prevention of the salt-sensitive component of hypertension and proteinuria in SHRSP with AUDA, a soluble epoxide hydrolase (sEH) inhibitor. 61st Annual High Blood Pressure Research Conference. Tucson Arizona, September 26-29, 2007. Accepted for oral presentation.

123. Li, J., M.A. Carroll, P.N. Chander, J.R. Falck, B. Hammock and C.T. Stier, Jr. A soluble epoxide hydrolase (sEH) inhibitor, AUDA, ameliorates end-organ damage in stroke-prone hypertensive rats (SHRSP). J. Hypertension 26 (suppl 1): S26, 2008.

124. Liclican, E., A.B. Doumad, J. Wang, J.C. McGiff, C.T. Stier, Jr. and M.A. Carroll. In vivo inhibition of the adenosine$_{2A}$ receptor-epoxyeicosatrienoic acids (A_{2A}R-EET) pathway renders Dahl salt-resistant rats salt-sensitive. 62st Annual High Blood Pressure Research Conference. Atlanta, Georgia, September 17-20, 2008. Accepted for late-breaking poster session.

125. Li, J., C.T. Stier, Jr., P.N. Chander, A.B. Doumad and M.A. Carroll, J.R. Protective role of epoxyeicosatrienoic acids (EETs) against renal damage in salt-loaded stroke-prone SHR (SHRSP). 63rd Annual High Blood Pressure Research Conference. Chicago, Illinois, September 23-26, 2009. Submitted for presentation.

126. Li, J., P.N. Chander, C.T. Stier and M.A. Carroll. Renal epoxygenase up-regulation ameliorates salt-sensitive hypertension and renal damage in SHRSP. American Society of Nephrology Renal Week 2009, San Diego, CA, October 27 – November 01, 2009. Accepted for poster presentation.

127. Chander, P.N., J. Li, M.A. Carroll and C.T. Stier. Soluble epoxide hydrolase (sEH) inhibitor, AUDA, markedly reduces thrombotic microangiopathy (TMA) in SHRSP. American Society of Nephrology Renal Week 2009, San Diego, CA, October 27 – November 01, 2009. Accepted for podium presentation.

128. Stier, C.T., Y-J. Kang, T. Manero and P.N. Chander. Niacin treatment markedly reduces proteinuria and glomerulosclerosis in rats with the metabolic syndrome. American Society of Nephrology Renal Week 2009, San Diego, CA, October 27 – November 01, 2009. Accepted for poster presentation.

129. Kang, Y-J., T. Manero, S. Levine, D. Owens, P.N. Chander and C.T. Stier, Jr. Niacin reduces renal damage and insulin insensitivity in Charles River Obese SHR (SHROB/cp): a model of the metabolic syndrome. Experimental Biology 2010, Anaheim, CA, April 24 – April 28, 2010. Accepted for poster presentation.

130. Battula, S., S. Hao, P.L. Pedraza, C.T. Stier, Jr. and N.R. Ferreri. Tumor necrosis factor-alpha (TNF) is an endogenous inhibitor of Na^+-K^+-2Cl^- cotransporter (NKCC2) in the medullary thick ascending limb (mTAL). 64th Annual High Blood Pressure Research Conference. Washington, D.C., September 23-26, 2010. Submitted for presentation.

National and International Presentations and Scientific Exhibitions
Charles T. Stier, Jr., Ph.D.

1978 Characterization of the natriuretic response to [7-glycine]-oxytocin in rats. Federation of American Societies for Experimental Biology meeting. Atlantic City, New Jersey.

1981 Bromocriptine effects on single nephron and whole kidney function in rats. Federation of American Societies for Experimental Biology meeting. Atlanta, Georgia.

1982 Diuretics and calcium excretion by the isolated canine kidney. American Society of Pharmacology and Experimental Therapeutics meeting. Louisville, Kentucky.

1983 Stereospecific intrarenal conversion of 5-hydroxytryptophan (5-HTP) to serotonin. Federation of American Societies for Experimental Biology meeting. Chicago, Illinois.

1984 Blood and urinary thromboxane in spontaneously hypertensive rats (SHR). Federation of American Societies for Experimental Biology meeting. St. Louis, Missouri.

Dopamine and serotonin as reciprocal intrarenal hormones. American Federation for Clinical Research meeting. Washington, D.C. (POSTER PRESENTATION).

1985 Thromboxane and the development of hypertension in spontaneously hypertensive rats (SHR). American Society of Pharmacology and Experimental Therapeutics meeting. Boston, Massachusetts. (POSTER PRESENTATION).

1986 Reduced dopamine formation in spontaneous hypertensive rats. American Federation for Clinical Research meeting. Washington, D.C.

Enhanced thromboxane formation by blood but not whole platelets from spontaneously hypertensive rats. American Federation for Clinical Research meeting. Washington, D.C. (POSTER PRESENTATION).

Natriuretic effect of 9α, 11β PGF in the rat. 6th International Conference on Prostaglandins and Related Compounds. Florence, Italy. (POSTER PRESENTATION).

Thromboxane in severe hypertension in stroke prone spontaneously hypertensive rats (SHRSP). American Society of Pharmacology and Experimental Therapeutics meeting. Baltimore, Maryland. (POSTER PRESENTATION).

1987 Preservation of kidney function and increased survival in stroke-prone SHR treated with enalapril. Federation of American Societies for Experimental Biology meeting. Washington, D.C.

1988 Increased survival and reduced proteinuria in salt-loaded stroke-prone spontaneously hypertensive rats (SHRSP) treated with captopril. Federation of American Societies for Experimental Biology meeting. Las Vagas, Nevada.

1989 Dose-dependent protective effects of SQ-29852 against stroke and hypertensive renal disease in stroke-prone SHR. American Society of Nephrology meeting. Washington, D.C. (POSTER PRESENTATION).

1990 Comparison of SQ-29852 and hydralazine in stroke-prone SHR (SHRSP) with severe hypertension and proteinuria. Federation of American Societies for Experimental Biology meeting. Washington, D.C. (POSTER PRESENTATION).

Loss of protection against stroke on withdrawal of ACE inhibitor therapy with SQ-29852 in salt-loaded stroke-prone SHR (SHRSP). June 28, 1990. 13th Scientific Meeting of the International Society of Hypertension. Montreal, Canada.

Dose-dependent protective effects of the ACE inhibitor SQ-29852 against stroke and hypertensive renal disease in stroke-prone SHR (SHRSP). 13th Scientific Meeting of the International Society of Hypertension. Montreal, Canada. (POSTER PRESENTATION).

Invited Seminar Speaker
Lederle Laboratories, Pearl River, New York. September 26, 1990 "ACE Inhibitors in the Prevention of Stroke".

Angiotensin II receptor antagonist DuP-753 prevents stroke and hypertensive renal disease in salt-loaded stroke-prone SHR. American Society of Nephrology meeting. Washington, D.C.

Invited Speaker
Eastern Hypertension Society, New York, New York. December 12, 1990. "Vascular Protection with ACE Inhibitors in Stroke-Prone SHR."

1991 Angiotensin II receptor antagonist DuP-753 prevents stroke and hypertensive renal disease in salt-loaded stroke-prone SHR. The Second International Symposium on ACE Inhibition. February 20, 1991. London, England.

Invited Seminar Speaker
St. George's University School of Medicine. Grenada, West Indies
March 15, 1991 "Stroke Prevention".

Visiting Professor of Pharmacology
St. George's Medical School, Grenada, West Indies
March 9, 1991 - March 16, 1991.

Role of the renin-angiotensin system in the pathology of salt-loaded stroke-prone SHR. American Heart Association High Blood Pressure Research Council Chicago, Illinois. (POSTER PRESENTATION).

1992 Role of the renin-angiotensin system in the pathology of stroke-prone SHR. Federation of American Societies for Experimental Biology Meeting. Symposium honoring Wilbur H. Sawyer at his retirement. Anaheim, California.

Cerebral vascular consequences (Stroke). Invited Speaker. Symposium, "Losartan: The First Angiotensin II Antagonist". St. Paul de Vence, France.

1993 Development of microangiopathic lesions (MAL) in Wistar Kyoto rats (WKY) upon chronic inhibition of nitric oxide (NO) synthesis by N (omega) nitro-L-arginine (L-NNA). American Society of Nephrology Meeting. Baltimore MD (POSTER PRESENTATION).

Protective action of beraprost sodium in stroke-prone SHR. American Society of Hypertension Meeting. New York, New York (POSTER PRESENTATION).

Endothelin receptor antagonism in stroke-prone SHR. Bristol-Myers Squibb. October 17, 1993. Princeton, New Jersey.

Role of the renin/angiotensin system and nitric oxide in the vascular pathology of stroke-prone SHR. Ciba-Geigy Corporation. December 1, 1993. Summit, New Jersey.

1994 Role of the renin-angiotensin system and nitric oxide in the vascular pathology of stroke-prone SHR. Invited Speaker. Pediatric Cardiology. March 23, 1994 Valhalla, New York.

Role of estrogen in the development of vascular pathology in female stroke-prone spontaneously hypertensive rats. Zeneca Pharmaceuticals Inc. May 9, 1994. Wilmington, Delaware.

Estrogen promotes the development of thrombotic microangiopathy (TMA) in female stroke-prone spontaneously hypertensive rats. American Society of Nephrology Meeting. October 25, 1994. Orlando, Florida (POSTER PRESENTATION).

1995 Role of renin-angiotensin system in the vascular pathology of stroke-prone SHR. Festschrift for Carl Gottschalk. April 6, 1995. Charlottesville, Virginia.

Pressor responsiveness to big-endothelin (Big-ET) in stroke-prone spontaneously hypertensive rats (SHRSP). Experimental Biology, Atlanta, GA (POSTER PRESENTATION).

Role of the renin-angiotensin system in the vascular pathology of stroke-prone SHR. Invited Speaker. Southern College of Optometry. November 16, 1995, Memphis, Tennessee.

Role of NO and endothelin in the vascular pathology of stroke-prone SHR. Invited Speaker. Departments of Physiology and Pharmacology, University of Tennessee. November 17, 1995. Memphis, Tennessee.

Role of NO and endothelin in the vascular pathology of stroke-prone SHR. Invited Speaker. UMDNJ School of Osteopathic Medicine. November 20, 1995, Stratford, New Jersey.

1996 Role of NO and endothelin in the vascular pathology of stroke-prone SHR. Invited Speaker. Department of Pathology. March 6, 1996 Valhalla, New York.

Angiotensin II in cardiovascular damage of SHRsp and its prevention by CEI. Laragh Day: All About Renin, The New York Hospital-Cornell Medical Center, May 14, 1996, New York, New York.

1998 Role of the renin-angiotensin aldosterone system in the development to stroke and renal vascular pathology. G.D. Searle & Co. March 18, 1998. Skokie, Illinois.

Aldosterone in renal and cerebrovascular disease. Invited Speaker. Neonatal Research Unit. March 25, 1998. Valhalla, New York.

Aldosterone Involvement in Stroke and Renal Disease. International Society of Hypertension. Selective Aldosterone Receptor Antagonists (SARAs) Symposium, June 7, 1998, Amsterdam, The Netherlands.

1999 Vascular protective effect of a selective aldosterone receptor antagonist in stroke-prone hypertensive rats. 25th Annual International Aldosterone Conference, San Diego, June 10 & 11, 1999.

Renal mechanisms and pathophysiology. Continuing Education Seminar on Renal Functional Disorders & Drug Therapy sponsored by the Empire State Pharmaceutical Society, Queens, New York, July 11, 1999.

Reactive oxygen species mediate proteinuria induced by inhibition of nitric oxide formation in stroke-prone SHR. American Society of Nephrology meeting. Miami Beach, Florida, November 5, 1999.

2000 Role of the renin-angiotensin-aldosterone system in renal microvascular injury in stroke-prone spontaneously hypertensive rats. Invited Lecture. University of Louisville, Department of Physiology and Biophysics, Louisville, KY, March 28th and 29th, 2000.

Diuretic Therapy and the Management of Renal Failure. Continuing Education Seminar on Renal Functional Disorders & Drug Therapy sponsored by Drug Experts, LaGuardia Crowne Plaza Hotel, New York, New York, June 11th, 2000.

Clues to the Vasculotoxic Action of Aldosterone. Pharmacia. Skokie, Illinois with videocast to St. Louis, Missouri, June 28th, 2000.

The role of aldosterone in models of vascular injury. Symposium 5 - Aldosterone: A new appreciation of its role in cardiac and vascular risk. Invited Speaker. 15th Annual International Interdisciplinary Conference on Hypertension and Related Risk Factors in Ethnic Populations (ISHIB-2000) El Conquestador Hotel and Resort, Las Croabas, Puerto Rico, July 15th to 18th, 2000.

Antioxidants reduce aldosterone-induced renal vascular injury in stroke-prone SHR. Poster Presentation at the International Society of Hypertension Meeting (ISH-2000): Chicago, Illinois, August 23-24th, 2000.

The role of aldosterone in cardiovascular end-organ damage. Heart Failure Society of America Special Symposium "SARA's in Heart Failure" Invited Speaker. 4th Annual Scientific Meeting of the Heart Failure Society of America, Boca Raton, Florida, September 11th, 2000.

Nitric oxide: historical perspectives, physiological functions and its connection with sildenafil (Viagra). Continuing Education Symposium for Pharmacists and Other Health Care Professionals sponsored by Drug Experts, Inc. "Erectile Dysfunction: The Sildenadil (Viagra) Experience and New Developments", Holiday Inn, JFK Airport, New York, New York, October 8th, 2000.

Potassium chloride intake provokes increases in plasma aldosterone and renal injury in non-sodium loaded stroke-prone spontaneously hypertensive rats (SHR). Oral Presentation at the American Society of Nephrology's 2000 Annual Meeting: Toronto, Canada, October 13th, 2000.

The role of aldosterone in stroke and renal damage in stroke-prone SHR. State-of-the-Art Lecture, American Heart Association Council for High Blood Pressure Research. Symposium "New Concepts on the Role of Aldosterone in Hypertension and Cardiovascular Disease" Invited

Speaker. 54th Annual Fall Conference and Scientific Sessions of the Council for High Blood Pressure Research, Omni Shoreham Hotel, Washington,D.C., October 27th, 2000.

2001 The Scientific Basis for Aldosterone as a Vascular Risk Hormone: Data From the Hypertensive Animal Model. Invited Lecture at the American Society of Hypertension symposium, "Aldosterone- A Common Link in Cardiac & Vascular Diseases". American Society of Hypertension, 16th Scientific Meeting and Exposition: San Francisco, California, May 15th -19th, 2001.

Estrogen promotes stroke and renal injury in stroke-prone hypertensive rats. Poster Presentation at The Endocrine Society's 83rd Annual Meeting (ENDO-2001): Denver, Colorado, June 20 th - 23rd, 2001.

Aldosterone-The Forgotten "A" in the RAAS. Symposium: Clinical Implications of Aldosterone. Chairman and Invited Speaker. 16th Annual International Interdisciplinary Conference on Hypertension and Related Risk Factors in Ethnic Populations (ISHIB-2001) The Mandalay Bay Resort & Casino, Las Vegas, Nevada, July 7th to 12th, 2001.

2002 Role of aldosterone in the development of stroke in genetically hypertensive rats. Circulation J. 66(Suppl I) 520, 2002. presented at The 66th Annual Scientific Meeting of the Japanese Circulation Society, Sapporo, Japan, April 24-26, 2002.

Aldosterone-induced renal osteopontin expression in stroke-prone hypertensive rats. Podium presentation at the 28th Annual International Aldosterone Conference, San Francisco, California, June 17 & 18, 2002.

Sodium transport antagonism reduces thrombotic microangiopathy in stroke-prone hypertensive rats (SHRSP) through a non-diuretic mechanism. Poster Presentation at the International Society of Hypertension Meeting (ISH-2002): Prague, Czech Republic, June 23-27th, 2002.

Sodium transport antagonism reduces thrombotic microangiopathy in stroke-prone hypertensive rats (SHRSP) through a non-diuretic mechanism. Podium Presentation at the 4th International Congress of Pathophysiology: Budapest, Hungary, June 30th, 2002.

The emerging role of aldosterone in cardiorenal disease. A Scientific Forum: The potential benefits of aldosterone blockade in preclinical and animal models. Chicago, Illinois, October 2nd, 2002.

The harmful effects of aldosterone in cardiorenal disease. Expert Forum: The emerging role of selective aldosterone blockade in the management of cardiorenal disease. American Society of Nephrology, Philadelphia, Pennsylvania, November 2nd, 2002.

Aldosterone as a mediator of end organ damage. Nephrology Grand Rounds. College of Physicians & Surgeons of Columbia University Renal Division. New York, New York, December 16th, 2002.

2003 "Aldosterone in cardiovascular disease". Cardiology Grand Rounds. Saint Vincent Catholic Medical Centers. Comprehensive Cardiology Institute: New York, New York, March 20th, 2003.

Renal Pathology and Toxicology, Poster Session: San Diego Convention Center. "Vasopeptidase inhibition affords greater renal protection and lowers plasma osteopontin levels at a low dose that

ACE inhibition in saline-drinking SHRSP". Experimental Biology 2003: San Diego, California, April 12th, 2003.

Aldosterone and not Ang II is associated with malignant nephrosclerosis and increase in-vivo expression of renal osteopontin in stroke-prone hypertensive rats. ALDO '03 International Symposium on Aldosterone, London, England, April 28th, 2003.

Inflammation and Cardiovascular Disease, Free Communication Session: New York Marriott Marquis. "Estrogen promotes microvascular pathology in female stroke-prone spontaneously hypertensive rats (SHRSP)". American Society of Hypertension, 18th Annual Scientific Meeting: New York, New York, May 13th, 2003.

"Aldosterone plays a pivotal role in the microvascular pathology of stroke-prone hypertensive rats". Endocrinology Rounds. SUNY Downstate Medical Center. Division of Endocrine, Diabetes and Heart: Brooklyn, New York, July 21st, 2003.

2004 Aldosterone and Cardiovascular Disease State of the Art Session, "Aldosterone and Cardiovascular Disease – Relationship to Blood Pressure". The American Society of Hypertension Nineteenth Annual Scientific Meeting and Exposition, New York Marriott Marquis, New York, New York, May 18-22, 2004.

"High potassium chloride intake provokes aldosterone-mediated proteinuria and renal injury in non-sodium-loaded stroke-prone spontaneously hypertensive rats (SHRSP)". Podium presentation at the 30th Annual International Aldosterone Conference, New Orleans, Louisiana, June 14 & 15, 2004.

"Review of anti-aldosterone cardiovascular and blood pressure effects". Expert Forum & Aldosterone Meeting, New York, New York, June 23, 2004.

2005 IUPS Renin-Angiotensin-Aldosterone (Endocrinology, Metabolism and Reproduction) Poster Session: San Diego Convention Center. "Enhanced end-organ protection in the absence of increased plasma potassium with combined mineralocorticoid receptor (MR) antagonist and amiloride therapy". Experimental Biology 2005: San Diego, California, April 2-6, 2005.

"Blood pressure independent effects of aldosterone". Session entitled, Breaking the Renin Angiotensin Aldosterone Axis. The National Kidney Foundation 2005 Spring Clinical Meetings, Marriott Wardman, Washington, DC, May 4-8, 2005.

"Cardiovascular benefits of mineralocorticoid receptor blockade beyond blood pressure lowering", Truman Memorial Veteran's Hospital and "Role of aldosterone in the end-organ damage of stroke-prone spontaneously hypertensive rats", University of Missouri, St. Louis, Missouri, July 12, 2005.

"End-organ benefits of mineralocorticoid receptor antagonism beyond blood pressure lowering", 16th Annual Vascular Biology and Hypertension Symposium, Destin, Florida, October 08, 2005.

2006 "Pharmacotherapy of hypertension and congestive heart failure". Continuing Education Symposium for Pharmacists and Other Health Care Professionals, sponsored by Drug Experts, Inc., Adria Hotel, New York, New York, March 19th, 2006.

"Cardiovascular consequences of long-term therapy with eplerenone in non-salt loaded stroke-

prone hypertensive rats". Podium presentation at the 32th Annual International Aldosterone Conference, Boston, Massachusetts, June 22 & 23, 2006.

2007 "Prevention of the salt-sensitive component of hypertension and proteinuria in stroke-prone SHR with the soluble epoxide hydrolase inhibitor AUDA". Renal Hemodynamics: Biomolecular Control Mechanisms Integrating Vascular & Tubular Function, sponsored by FASEB Summer Research Conferences, Vermont Academy, Vermont, July 7th - 12th, 2007.

2008 "A soluble epoxide hydrolase (sEH) inhibitor, AUDA, ameliorates end-organ damage in stroke-prone hypertensive rats (SHRSP)". OS23/2 International Society of Hypertension Meeting, Berlin Convention Center, Berlin, Germany, June 14-19, 2008.

American Heart Association Scientific Sessions 2008, Oral Abstract Session Moderator, Session Title: "Hypertension: Experimental – Diabetes, Renal Sodium Retention, Angiotensin II and Cardiovascular Responses", 9:00AM – 12:00 noon, New Orleans Convention Center, New Orleans, Louisiana, November 8-12, 2008.

2009 "The effectors modulating the effects of the pro-fibrotic and pro-inflammatory effects of aldosterone: A pathophysiological Update-2009". Session entitled, Aldosterone Axis. Inter-American Society of Hypertension, Belo Horizonte, Brazil, August 4-8, 2009.

"Comparative pathologic studies of WKY, SHR, SHRSP and SHROB". Charles River Laboratories Research Investigators Symposium, Chicago, Illinois, September 22, 2009.

"Use of radiotelemetry in hypertension research". Data Sciences Users Group Meeting, Harvard School of Public Health, Boston, Massachusetts, September 30, 2009.

"Protection against renal damage in spontaneously hypertensive rats that are either stroke-prone (SHRSP) or Obese (SHROB)". Boehringer Ingelheim Pharmaceuticals, Ridgefield, Connecticut, October 21, 2009.

"Renal epoxygenase up-regulation ameliorates salt-sensitive hypertension and renal damage in SHRSP". American Society of Nephrology Renal Week 2009, San Diego, California, October 29, 2009.

"Niacin treatment markedly reduces proteinuria and glomerulosclerosis in rats with the metabolic syndrome". American Society of Nephrology Renal Week 2009, San Diego, California, October 30, 2009.

"Soluble epoxide hydrolase (sEH) inhibitor, AUDA, markedly reduces thrombotic microangiopathy (TMA) in SHRSP". American Society of Nephrology Renal Week 2009, San Diego, California, October 30, 2009.

Ph.D. Thesis Committees

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Anand Lokhkar (2010)

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Alafuro Oruene (2000-2001)

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Saleem Ahmad (1988)
Fanming Lin (1989)
Vincent DiPippo (1990)
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Editorial; Ad Hoc Reviewer

American Journal of Kidney Disease
American Journal of Hypertension
American Journal of Physiology
Archives of Medical Research
Biochimica et Biophysica Acta
Canadian Journal of Physiology & Pharmacology
Cardiovascular Research
Cardiovascular Drug Reviews
Clinical Nephrology
Clinical Science
Current Medicinal Chemistry-Cardiovascular and Hematological Agents
Drugs
Endocrinology
European Journal of Pharmacology
Experimental Nephrology
Heart
Hypertension
Journal of the American Society of Nephrology
Journal of Cardiovascular Pharmacology
Journal of Clinical Endocrinology and Metabolism
Journal of Clinical Investigation
Journal of Hypertension
Journal of Neuroendocrinology
Kidney International
Lancet
Molecular and Cellular Endocrinology
Pharmacological Research
Prostaglandins
Science
Stroke

Expert Fee Schedule & Prior Testimony

Fee Schedule

Dr. Stier charges $300 per hour for record review, drafting of reports, preparation of and giving deposition testimony. He charges $3,000 for testimony at trial.

Prior Testimony

Date	Name of Case	Court No.	Docket No.:
08/31/09	Composto vs Chandler et al. (Trial testimony)	Ulster County. New York State Supreme Court	3030/04